INITIAL CLINICAL EXPERIENCE WITH A NEW PERMANENT MECHANICAL AUXILIARY VENTRICLE: THE DYNAMIC AORTIC PATCH

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Developments in mechanical circulatory support, diagnostic cardiology, and roentgenology during the last 20 yr have spurred exploration of experimental approaches to the amelioration of advanced atherosclerotic heart disease and its complications, hypertensive cardiovascular disease, and cardiomyopathies of various etiologies. Among these methods are coronary revascularization and endarterectomy, resection of ventricular aneurysms and repair of valvular lesions secondary to acute myocardial infarction, and cardiac transplantation.

Another approach, in-series mechanical circulatory assistance, appears promising in the management of both chronic and acute forms of medically intractable, progressive left ventricular failure. Based on the experimental demonstration that retarding the arterial pressure pulse to the diastolic phase of the cardiac cycle leads to increase in coronary artery blood flow[1], several techniques of diastolic augmentation have been developed[2,3]. Of these, the most effective in support of the acutely failing ventricle appears to be intraaortic phase-shift balloon pumping[4-7]. In this method, a flexible polyurethane pumping chamber in the descending thoracic aorta is expanded during ventricular diastole and deflated at the beginning of systole[8]. Beneficial effects on hemodynamic parameters and restoration of the acutely failing circulation to a stable operating point as a result of balloon pumping have been documented in laboratory studies by a number of investigators[3,7]. Clinical trials have corroborated the experimental observations and accumulating data on the effectiveness, low risk, and simplicity of the method suggest that it may become the treatment of choice in cardiogenic shock secondary to acute myocardial infarction[7,9].

The chronically failing ventricle can also be effectively supported by means of diastolic augmentation. In 1966, implantations of a U-shaped mechanical auxiliary ventricle demonstrated beneficial hemodynamic and physiologic changes in 2 patients with chronic left ventricular failure. In the second case substantial hemodynamic improvement was obtained during the first 10 days of the postoperative course, suggesting the feasibility of permanent in-series circulatory assistance. However, the applicability of this system was restricted by the thromboembolic potential of the prosthesis[2,10,11].

To overcome this limitation, a new auxiliary ventricle, the dynamic aortic patch[12], was developed. Its operating principle and hemodynamic effects correspond very closely with those of the intraaortic balloon pump[7,12]. Its initial utilization in a patient with advanced chronic left ventricular failure is described in this report.

PROSTHESIS

The dynamic aortic patch (Figure 1) is an ellipsoidal silicone rubber pumping chamber approximately 15 cm in length and 3 cm in width. Fully inflated with air or oxygen, its volume is approximately 33 cc. The pumping chamber is implanted longitudinally in the lateral wall of the descending thoracic aorta between the origin of the left subclavian artery and the diaphragm (Figure 2). The intravascular surface of the pumping chamber is covered with dacron velour backed by a conductive polyurethane[13-16]; plain dacron is used to cover the extravascular surface and also the 0.25" conduit leading from the dynamic aortic patch to a transcutaneous connector (Figure 3), which in turn is joined to an extracorporeal pressurized gas supply and driving unit.

To obtain phase-shift pumping with the dynamic aortic patch, 3 driving systems are available. For bedside use the AC-powered unit developed by our group for intraaortic balloon pumping is operated with pressurized air or oxygen. To provide a mobile driving system, this unit is mounted in a wheel chair. Power is provided by rechargeable batteries. The compressed air supply is provided by 2 scuba tanks with capacity for 6 hr operation. The third driving system is vest-mounted and consists of a continuously operating miniature compressor which maintains air pressure in a 500 cc reservoir. Power is provided by rechargeable batteries with a capacity of approximately 3 hr. In each of these driving systems, the R wave of the electrocardiogram is used to trigger the operation of a solenoid valve, which inflates and deflates the pumping chamber according to the requirements for phase-shift pumping[8].

CASE REPORT

On July 30, 1971 a 63 year old male with symptoms of advanced chronic congestive heart failure refractory to pharmacologic therapy was transferred from another hospital. He was bedridden because of weakness, severe dyspnea, and leg edema.

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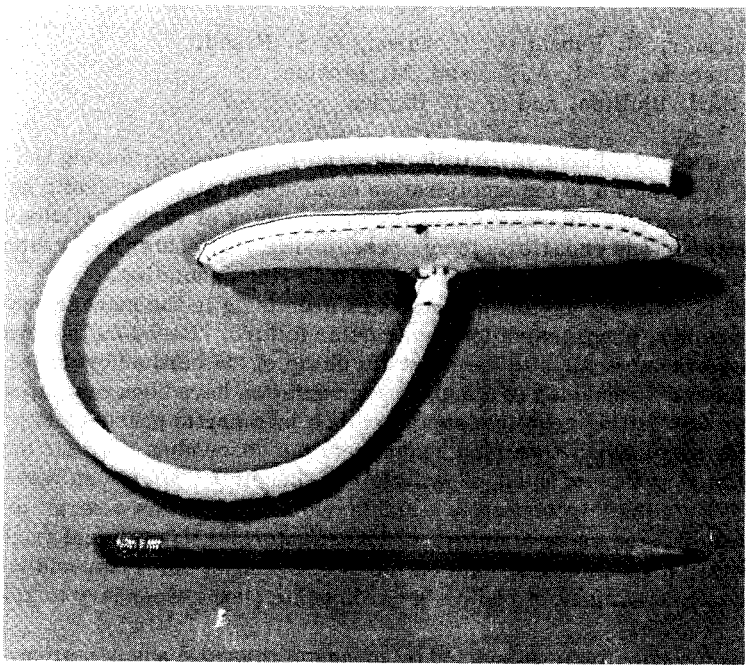

Figure 1. Dynamic aortic patch.

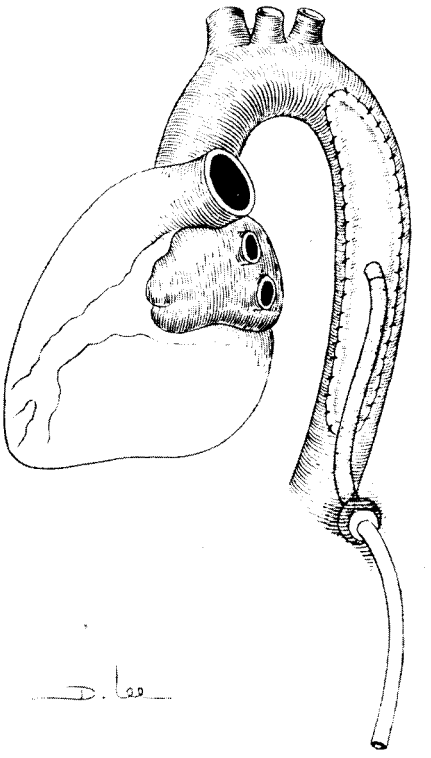

Figure 2. Schematic illustration showing intra-aortic location of dynamic aortic patch. (Reproduced from Kantrowitz, et al.[7] by courtesy of Transplant. Proc.)

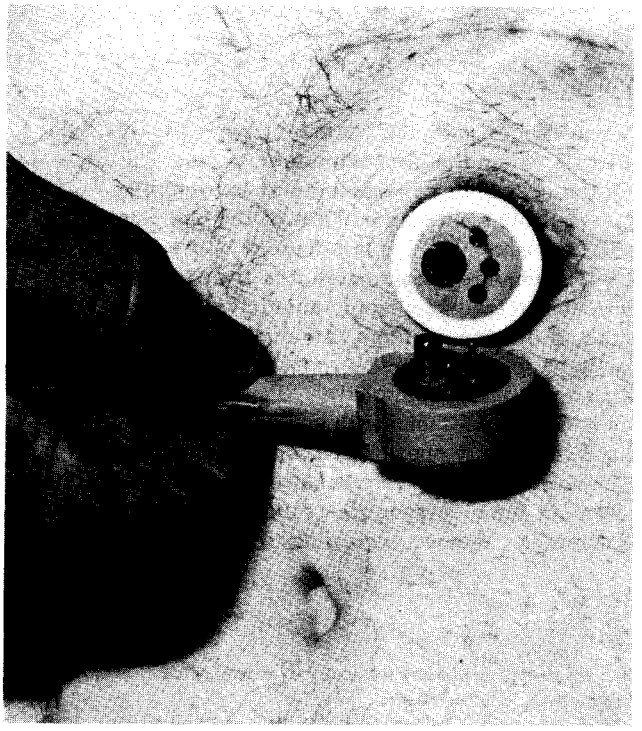

Figure 3. Transcutaneous connector implanted in the patient.

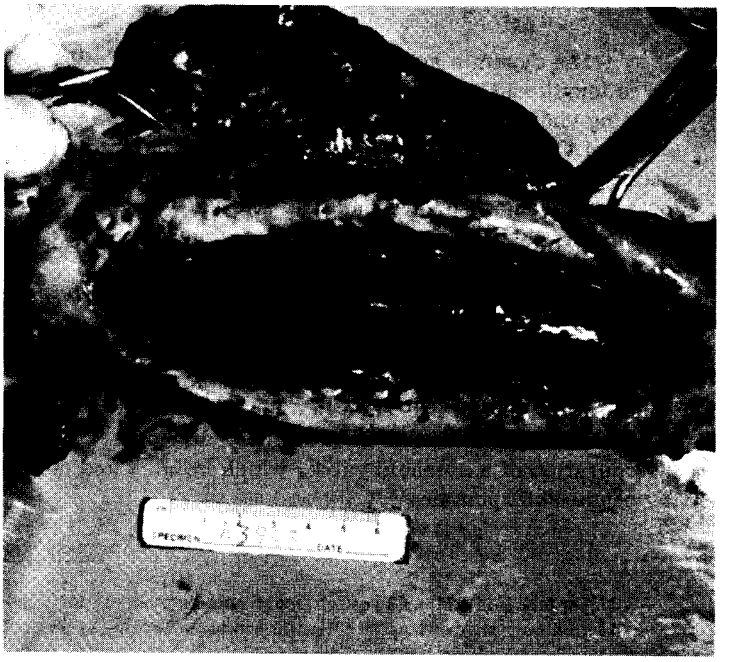

Figure 4. Intravascular surface of dynamic aortic patch at postmortem examination. Pumping chamber has been inflated. A stable fibrin layer covers the entire surface.

Symptoms had first occurred in 1966, when he complained of difficulty in breathing and a productive cough. Left heart failure was diagnosed, and treatment included digitalis and diuretics. This conservative regimen provided moderate relief until 1969, when the patient complained of palpitations. An EKG revealed complete left bundle-branch block, first degree atrioventricular block, and frequent premature ventricular contractions. A glucose tolerance test indicated borderline diabetes mellitus. The diabetes was well controlled by dietary measures, but the patient's cardiovascular status steadily deteriorated during 1970 despite treatment with large doses of furosemide, spironolactone, thiazides, digoxin, and quinidine. In addition, propranolol was given in small doses for control of arrhythmias. In May of 1970 he was hospitalized for persistent cough, paroxysmal nocturnal dyspnea, and retrosternal chest pain associated with activity.

Right and left cardiac catheterization, coronary arteriography, and left ventriculography were performed elsewhere on May 11, 1971. The results (Table I) disclosed severe left and right ventricular failure and severe pulmonary hypertension. The left ventricle was enlarged, dilated,

TABLE I

RESULTS OF CATHETERIZATION BEFORE AND AFTER IMPLANTATION OF DYNAMIC AORTIC PATCH

		LV*	PCW	MPA	CI	ΔEh
Mt. Carmel	(5-11-71)	90/15	(29)	64/22 (37)	2.2	
Sinai	(8-4-71)					
Control		80/24	(28)	50/23 (33)	2.5	- 2.92
Balloon ON (2 hours)		86/10	(14)	24/10 (15)	3.0	+ 2.22
Postop. 5 wk	(9-16-71)					
Patch Booster ON		104/16	(14)	34/18 (24)	4.32	+ 8.8
Patch Booster OFF (90 min)		100/16	(18)	38/21 (25)	3.79	+ 8.1
Patch Booster OFF Post Cine		100/16	(24)	43/21 (29)	3.79	- 2.0
Patch Booster ON (10 min)		93/10	(14)	32/10 (18)	4.26	+ 1.07

*LV = Left ventricle: peak/end-diastolic pressure (mm Hg)
MPA = Main pulmonary artery: peak systolic/end-diastolic and (mean) pressures (mm Hg)
PCW = Pulmonary capillary wedge pressure--mean (mm Hg)
CI = Cardiac index ($L/min/M^2$)
ΔEh = Myocardial oxidation-reduction potential (mV) reflecting lactate extraction (positive) or lactate production (negative).

$$\Delta Eh\ (mV) = 30.7 \times \log \frac{La_1 \times Py_2}{La_2 \times Py_1}$$

$La_1 + Py_1$ = the concentration of lactate and pyruvate in arterial blood
$La_2 + Py_2$ = the concentration of lactate and pyruvate in coronary sinus blood
30.7 = Constant

and contracted poorly; its ejection fraction was estimated at 30%. There was 1+ mitral regurgitation and more than 70% occlusion of the main right, main left, and anterior descending coronary arteries. Because of the severity of the cardiac symptoms in the absence of an anginal syndrome or a clear-cut history of myocardial infarction, the coronary artery disease was considered inoperable.

On May 19, 1971, a permanent transvenous pacemaker was implanted in the right ventricle to control premature ventricular contractions. Subsequently, the patient was hospitalized 5 times because of pulmonary and severe peripheral edema. During the preceding 10 months, the transverse diameter of the heart had increased from 165 to 190 mm. Chest X-rays also disclosed pulmonary vascular congestion, left pleural effusion, and persistent Kerley B lines in both lung bases.

At the time of transfer to our hospital, the patient's blood pressure was 110/70 mm Hg. The pulse rate, controlled by the fixed-rate pacemaker, was 92/min. His musculature was generally wasted, particularly in the region of the shoulder girdle and in the extremities. The abdomen was slightly scaphoid. There was slight neck vein distention at 30°, and a positive hepatojugular reflux. Fine, moist rales were heard at each lung base. The left border of cardiac dullness was in the sixth intercostal space in the anterior axillary line. A prominent apical impulse was noted with a loud left ventricular S_3 gallop and a soft Grade II/VI pansystolic apical murmur. The liver was tender and palpable 3 cm below the right costal margin. The peripheral pulses were normal and there was 1+ pretibial edema. Neurologic examination was normal except for a decreased sensory perception to pinprick in the lower extremities. Psychological testing revealed mild organic brain syndrome with some depression and loss of recent and remote memory.

Thorough laboratory evaluation revealed moderately severe liver impairment, mild reduction in renal function, and normal hematologic, endocrine, and GI systems. The clinical diagnoses included ischemic cardiomyopathy and atherosclerotic heart disease, New York Heart Classification IV-E; diabetes mellitus; chronic passive congestion of the liver with probable cardiac cirrhosis; mild organic brain syndrome, and diabetic peripheral neuropathy.

Since the dynamic aortic patch and the intraaortic balloon pump correspond closely in stroke volume, intraaortic location, and operating principle, a subject's response to balloon pumping provides a basis for predicting the hemodynamic effects of the permanently implanted system. Accordingly, on August 4, balloon pumping* was performed while the patient was in the cardiac catheterization laboratory. The results are presented

*Equipment manufactured by the Milton Roy Company, St. Petersburg, Florida.

in Table I. Before initiation of cardiac assistance, the patient was in marked left ventricular failure. The left ventricular end-diastolic pressure was 24 mm Hg and the pulmonary artery and pulmonary capillary wedge pressures were also elevated. Lactate and pyruvate studies indicated anaerobic myocardial metabolism.

After approximately 2 hr balloon pumping there was substantial improvement in these hemodynamic and metabolic determinations. Interruption of balloon pumping was followed promptly by reappearance of signs of failure, but reinitiation of cardiac assistance once more brought about improvement. Because of the favorable responses to balloon pumping, the patient was considered an appropriate candidate for patch booster implantation.

Surgical procedure. On August 10, 1971 the dynamic aortic patch was implanted according to the technique developed in animal studies in our laboratory (12). Balloon pumping had been initiated several hours prior to the procedure and was in progress when the patient was transported to the operating room. The thoracic cavity was entered through a left thoracotomy in the fourth intercostal space. Balloon pumping was discontinued when total cardiopulmonary bypass had been established. The descending thoracic aorta was dissected free and clamped at the level of the left subclavian artery and distally at the level of the diaphragm. Six pairs of intercostal arteries were also occluded temporarily. A 15 cm incision was then made on the lateral surface of the aorta between the proximal and distal clamps. The partial artificial heart was sewn into the vessel wall using a double row of continous everting sutures reinforced with dacron felt. The gas conduit was led out of the chest through the transcutaneous connector, which was implanted in the left hypogastric region (Figure 3), and then connected to the driving unit.

A pacing electrode was sutured to the left atrium and a fixed-rate pacemaker* was implanted subcutaneously in the left chest wall; the rate was set at 95 / min. The previously implanted transvenous pacemaker powerpack was removed but the catheter was left in place. Electrodes were sutured to the apex of the left ventricle with the leads brought out through the skin connector, where an electrical connection was provided (Figure 3). These electrodes carried the electrocardiographic signal to the driving unit. The chest was closed in routine fashion. Initially, during weaning from bypass, the pulmonary artery and central venous pressurers were elevated but returned to normal levels upon activation of the dynamic aortic patch and administration of small doses of isoproterenol hydrochloride.

Postoperative course. The patch booster was activated continually for the first 2 postoperative days; thereafter, it was used intermittently. During the first several days, the patient's hemodynamic status stabilized, with pulmonary artery pressure in the normal range. Pulmonary capillary wedge pressure, however, was elevated to 13-15 mm Hg. No evidence of right ventricular failure was present. During the first week, atrial fibrillation occurred intermittently. With increased doses of digitalis, the atrial rate was reduced and the patient remained in atrial pacemaker rhythm, with only occasional premature ventricular contractions.

Except for this complication and a shallow dehiscence of the chest incision, which was repaired under local anesthesia, the patient made good progress during the remainder of this hospitalization. His exercise tolerance gradually increased, permitting him eventually to walk several hundred yards without assistance. Hematologic studies, which included frequent determination of haptoglobin, urine hemosiderin, and microscopic red cell morphology, showed no evidence of hemolysis. Scanning electron microscopy with utilization of Nomarski optics as well as transmission electron microscopy demonstrated no abnormalities of red cell or platelet morphology or function. The cardiac transverse diameter decreased from 190 mm preoperatively to 160 mm 21 days postoperatively (essentially normal). The patient was on occasion agitated and depressed, possibly as a reflection of his chronic organic brain syndrome, and his appetite was often poor. These emotional manifestations responded to psychiatric management. The patient's nutrition, however, remained marginal.

Complete cardiac catherterization was repeated 5 wks postoperatively; the results are summarized in Table I. With the partial artificial heart activated, good hemodynamic compensation with marked increase in cardiac index was observed. There were minimal signs of failure and myocardial metabolism was aerobic. Whereas preoperatively, the myocardial oxidation-reduction potential during balloon pumping had been +2.2 mv, on this occasion it was +8.8 mv with the patch booster activated, indicating increased lactate extraction. Observation for 90 min during which the assist device was inoperative did not disclose significant deterioration of hemodynamic parameters. This indicated considerable improvement in the patient's cardiac reserve, as compared with the preoperative period, when significant deterioration had followed interruption of balloon pumping. However, injection of cineangiographic contrast material caused deterioration of hemodynamic parameters; myocardial metabolism reversed to an anaerobic state, and myocardial contractility, as reflected by left ventricular dp/dt and work index, gradually decreased. When cardiac assistance was resumed, these parameters returned toward their previous values within 10 min.

The patient was discharged on September 18. At home his schedule usually consisted of 2 hr support by the partial artificial heart, followed by 2 hr during which it was inactive. For the first 2 wk, his condition was relatively good. Fourteen days after his discharge, however, a low-grade fever (99° to 100°F) was noted.

On October 4, the patient was readmitted because of the fever, anorexia, malaise, and a 10 lb weight loss. Blood, sputum, and urine cultures had been negative prior to admission, but the white blood cell count and sedimentation rate were now elevated. Cardiac fluorscopy and 4-view X-ray study of the chest demonstrated an air-filled loculated cavity about 12 x 7 cm in size with a fluid level adjacent to the extravascular surface of the aortic patch.

On physical examination, the patient's temperature was 102.4°F, and muscle wasting and intermittent confusion were noted. A small amount of superficial yellowish exudate surrounded the skin button. The fever, clinical syndrome of weight loss, and malaise indicated intrathoracic infection, probably associated with the pneumatocele. From the paramediastinal cavity 100 cc of thick, brown purulent fluid and air were aspirated, and cultures revealed coagulase-positive, methicillin-sensitive Staphylococcus aureus. Methicillin was accordingly given intravenously and by instillation into the thoracic cavity through a catheter. The fluid within the sac was resorbed, but the patient continued to be lethargic and occasionally confused and febrile. With intermittent cardiac assistance (2 hr "on" and 2 hr "off"), his cardiac status was relatively stable.

On October 13, 64 days following implantation of the patch, the posterior half of the eighth rib was resected and the empyema cavity was drained under local anesthesia. From October 20 to October 28, moderate congestive failure was present, and there were several episodes of ventricular tachycardia and fibrillation. Bretylium Tosylate and other medications were used to control the arrhythmias. Fever persisted in spite of therapy.

Intermittent loss of capture of the atrial pacemaker was noted on October 29. The central venous pressure rose to 18-20 cm H_2O, the mean pulmonary artery pressure to 30-35 mm Hg, and urine output began to decline. These findings reflected increasing right as well as left heart failure. Because the patient remained febrile, anorectic, and in severe negative nitrogen balance, a tube gastrostomy was performed under local anesthesia to facilitate alimentation. Nevertheless, the BUN and creatinine began to rise, urine output continued to fall and the patient gradually became edematous and more dyspneic. Severe renal failure developed. Peritoneal dialysis, started on November 6, reduced the extent of the peripheral edema but pulmonary congestion persisted. During the week of dialysis, intermittent second-degree AV block of the Wenkebach type occurred; an occasional failure of atrial pacemaker capture was also noted. The frequency of premature ventricular contractions declined. On November 14, following an episode of ventricular tachycardia and fibrillation, a period of asystole occurred that could not be managed in spite of

* Manufactured by the General Electric Company, Milwaukee, Wisconsin.

ventricular pacing and other treatment. The patient died on the ninety-sixth postoperative day. The partial artificial heart had provided hemodynamic support virtually until his demise.

Autopsy findings. The dynamic aortic patch was intact. Its intravascular surface was covered with a glistening layer of fibrin. In most areas this layer was 1 to 2 mm thick but in some peripheral areas it was 5 mm. Function of the patch, as demonstrated by injecting 35 cc of air, was also preserved (Figure 4). Microscopically, the layer of fibrin had entrapped within its meshwork a few blood cells and had penetrated into the dacron fabric, between the refractile synthetic fibers (Figure 5). Organization had not yet occurred.

There were no indications of thromboembolization in any of the tissues examined, including brain and extremities.

The air tube was fixed in its course by surrounding tissue densely adherent to the dacron velour wrapped around it. On microscopic examination the interstices of the synthetic fabric were invaded by foreign body giant cells and fibroblasts (Figure 6). The region of the skin button, the source of a yellowish exudate in the postoperative period, was clear of infection. In contrast, the adjacent electrode catheter, also originating in the skin button, was involved in an infectious process. Pus had accumulated in the tissue surrounding the subcutaneous pacemaker powerpack located in the upper left chest. This purulent cellulitis extended from the pacemaker powerpack around the electrode catheter to the site of implantation of these electrodes. In addition, there was empyema involving all layers of the thoracotomy wound from the skin to the adventitial surface of the aorta in the region of the patch; an acute bilateral bronchopneumonia and septicemia; and a purulent peritonitis due to a leak about the gastrostomy. Cultures of these sites grew out mixed enteric organisms.

Autopsy also disclosed the patient's underlying severe arteriosclerotic heart disease. The heart was enlarged to 558 Gm. Old infarcts were present in the anterior, posterior, and septal regions of the left ventricle. On the anterolateral aspect of the heart was an area of hemorrhage and coagulation necrosis, indicating infarction within the last 48 to 72 hr. There were signs of congestive heart failure including acute and chronic passive congestion of the liver and lungs. The kidneys were enlarged and there was acute tubular degeneration.

COMMENT

The experience in this case suggests that a partial artificial heart which is suitable for intermittent use and relatively simple in construction, implantation procedure, and operation effectively supported the circulation of a patient with medically intractable, progressive left ventricular failure (Figure 7). Signs of left ventricular failure were rarely present while the patch booster was active. Probably as a result of cardiac assistance, the patient's functional capacity increased markedly during the first several postoperative weeks and his heart size decreased to the normal range.

In addition, the experience with this patient strongly suggests that encouraging progress has been made toward solution of the problem of an artificial intravascular surface for cardiac assist prostheses. Previously, our group had studied a U-shaped mechanical auxiliary ventricle[10,11]. This system, in which a silicone rubber inner surface was in direct contact with the blood, was implanted across the aortic arch. In the second clinical trial of this system, a good hemodynamic response was obtained, but the patient died as a result of embolization on the tenth postoperative day from a thrombus occupying the distal limb of the prosthesis. It was our impression, based on findings in animal experiments[11], that the junction between the silicone rubber pumping chamber and the dacron efferent limb of the prosthesis was the site at which thrombus formation began. In addition, the flow pattern when the prosthesis was inactive was nonphysiologic. These factors may have contributed to formation of an unstable thrombus in this case.

The dynamic aortic patch was designed to avoid these difficulties. When the device is inactive, there is minimal interference with the physiologic pattern of blood flow. In addition, it has an intravascular surface which is expected to favor formation of a stable pseudointimal layer.

In developing this material, we adapted the approach of Sharp and his colleagues[13-16]. In a series of studies these investigators defined some of the physical properties of a vascular prosthesis on which a thin, stable fibrin layer forms in vivo. Their data indicated that dacron velour backed by a conductive polyurethane meets these requirements. Confirmation of this finding was obtained in long-term vascular replacement experiments.

Our own studies using a similar material for the intravascular surface of the aortic patch in more than 150 dogs indicated that within 4 to 6 months after implantation, an organized pseudointimal layer develops, completely covering the blood interface of the prosthesis[7]; a detailed report of these experiments is in preparation. In these studies only one instance of possible embolization was discovered (an infarct 0.1 mm in diameter in the renal cortex of one animal).

The clinical experience with the dynamic aortic patch appears to confirm that dacron velour backed by conductive polyurethane promotes development of a stable fibrin layer in the human. Although the partial artificial heart had been used for 3 months on a daily basis, there was no evidence of embolization in any of the tissues examined. Ninety-six days after implantation of the dynamic aortic patch the synthetic material was completely covered by a stable fibrin layer which had a smooth, glistening surface, not subject to fragmentation and embolization. In time, such a surface would be expected to re-endothelialize.

Probably the major factor in this patient's death was extensive infection complicated by renal shutdown. The infection may have originated at the time of breakdown of the skin incision. Alternatively, the primary factor in the infection may have been diffusion of gas across the silicone rubber pumping chamber into the periaortic region. Oxygen was used to inflate the dynamic aortic patch while the patient was in the hospital and might have provided an excellent medium for bacterial growth. Experiments designed to investigate this possibility are in progress. Methods of rendering the pumping chamber impermeable are also being studied.

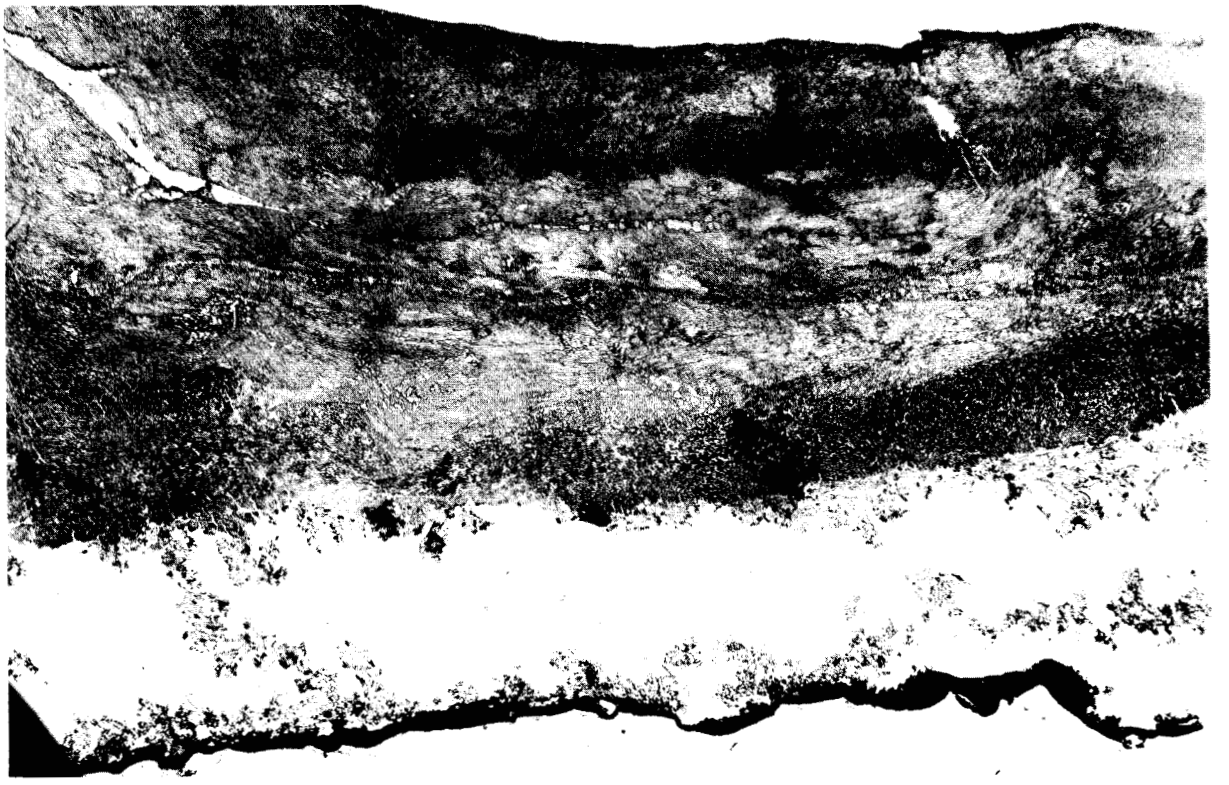

Figure 5. Photomicrograph of section through intravascular surface of aortic patch showing fibrin layer (above) and dacron velour backed by conductive polyurethane. Fibrin has penetrated dacron velour. (Clear space between fibrin and dacron is an artifact.) (X 25)

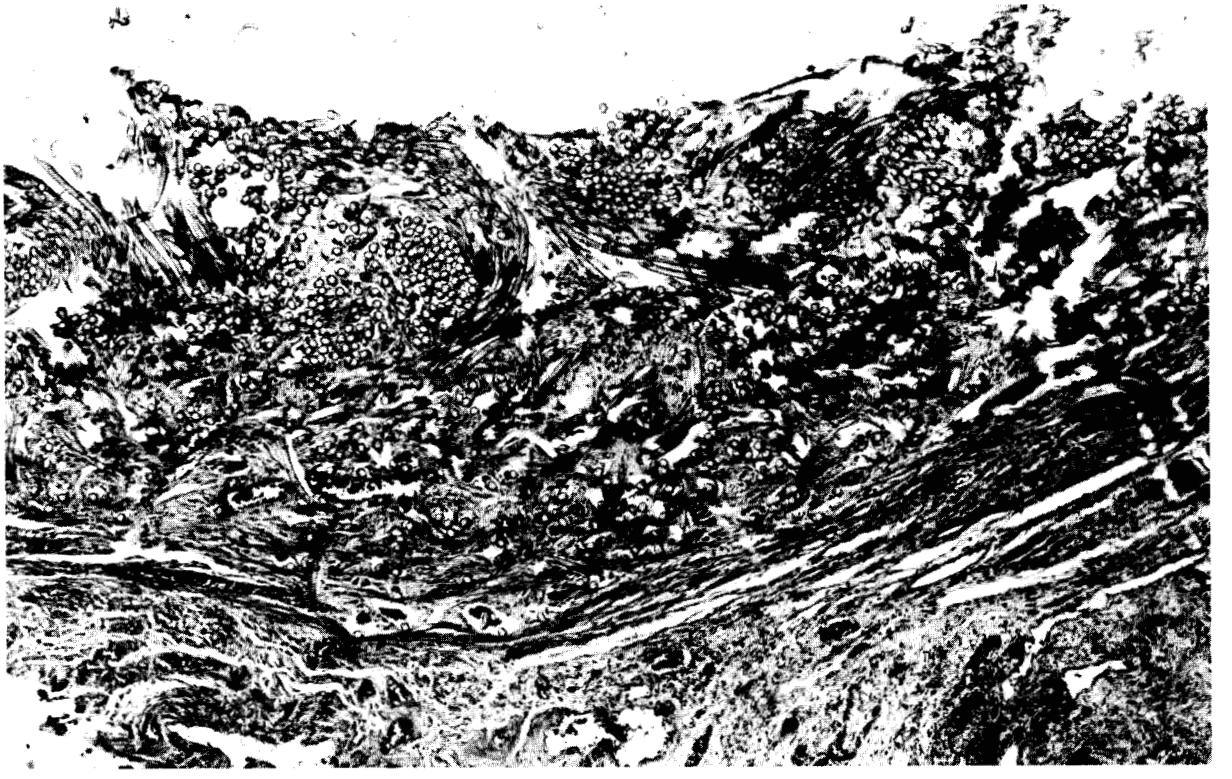

Figure 6. Photomicrograph of section through dacron velour surrounding gas conduit showing fibroblasts and foreign body giant cells invading interstices between synthetic fibers. (X 75)

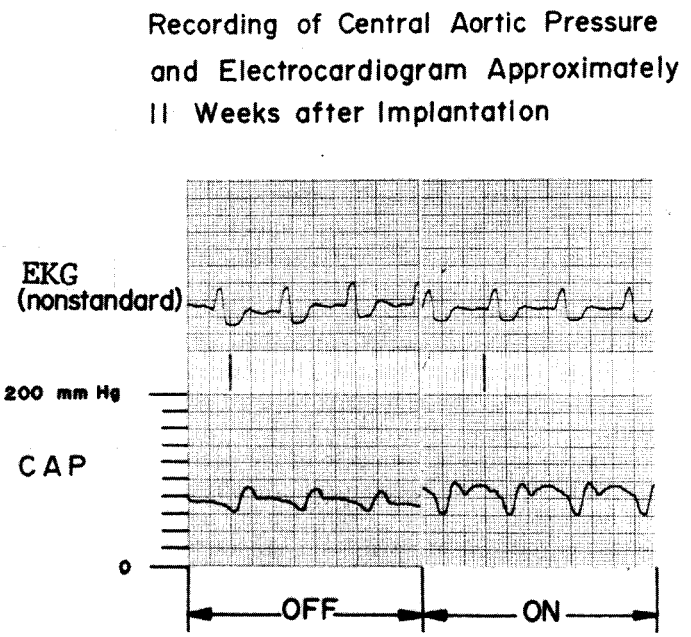

Figure 7. Recording of central aortic pressure and electrocardiogram approximately 11 wk after implantation.

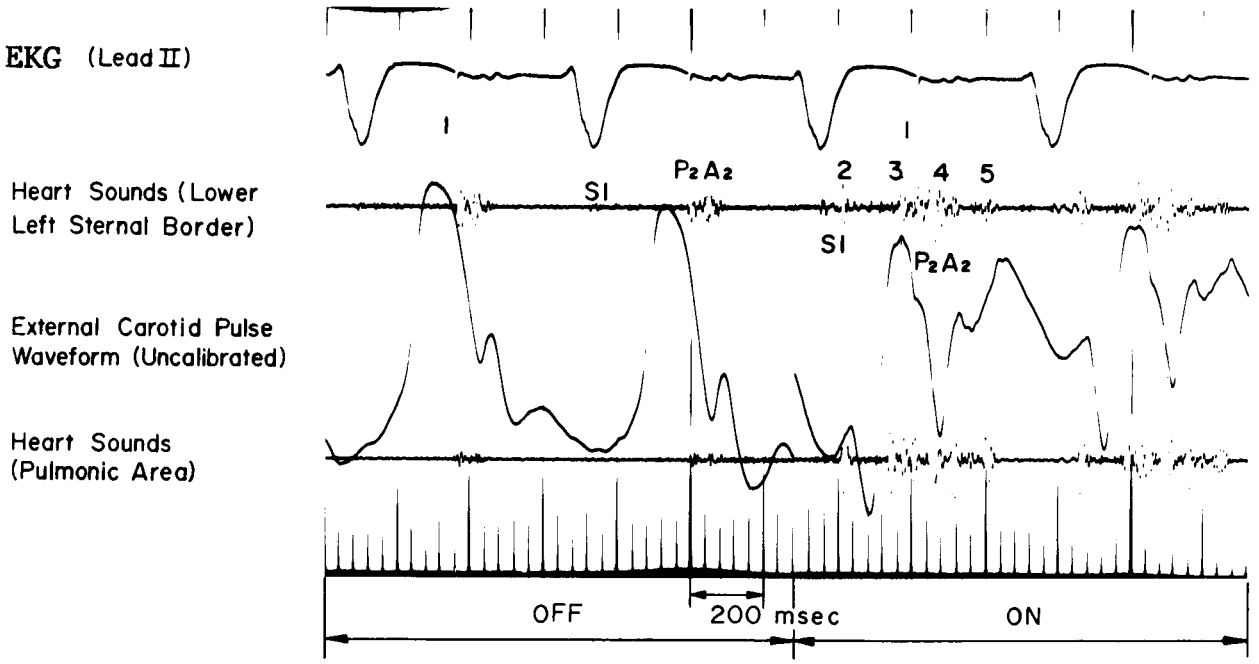

Figure 8. Recording of external carotid pulse waveforms, heart sounds, and electrocardiogram with patch booster active and inactive 6 wk after implantation. Key: 1 = pacemaker spike; 2 = initiation of patch deflation; 3 = patch completely deflated; 4 = initiation of patch inflation; 5 = patch completely inflated.

Although the infection was widespread, it did not involve the gas conduit. Evidently the dacron velour covering the air tube allowed obliteration of the potential space between this prosthetic structure and the surrounding tissue. In contrast, tissues surrounding the adjacent electrode catheter, which was not covered with dacron velour, did become infected.

The transcutaneous connector, clinically the site of a slight exudate during a short period, was not involved by infection at autopsy. This device, which was based on the design developed by the Epoxylite Corporation under the Artificial Heart Contract Program, evidently effectively inhibited passage of infectious agents, as reported by others[17]. McDonald, et al[18] greatly reduced infection in patients undergoing arteriovenous shunts and permanent peritoneal dialysis by providing the arteriovenous cannulas and dialysis catheter with a dacron cloth skirt implanted subcutaneously. They also found extensive fibroblastic reaction around the dacron skirt, which enhanced tissue fixation and reduced infection. The silicone rubber pumping chamber, which was estimated to have sustained 13 million inflations, showed no gross indications of wear or fatigue.

The 3 driving units functioned effectively throughout the patient's postoperative course. However, he did not require continuous cardiac support, for the most part he used the bedside unit, disconnecting himself when he wished to be free to move about.

The adjustment of the inflation and deflation cycle of the partial artificial heart was established at surgery and verified 5.5 wk later in the catheterization laboratory. No difficulties were encountered in maintaining correct timing, but this was to be expected, since cardiac pacing was employed. The possibility of adjusting the timing on the basis of the external carotid pulse wave form and the heart sounds as well as the assist device sounds (Figure 8) was evaluated. This seemed to be a feasible method of adjusting the timing but is currently being further studied.

SUMMARY AND CONCLUSIONS

A dynamic aortic patch, a permanent mechanical auxiliary ventricle, was implanted in a 63-year-old man with advanced left ventricular failure refractory to medical therapy. During a 96 day postoperative course, the prosthesis was used intermittently every day for hemodynamic support. Except for arrhythmias during the first week and a shallow dehiscence of the chest incision, the patient recovered from the surgical procedure without major complication. His condition improved markedly. Whereas preoperatively he had been bedridden as a result of weakness and dyspnea, postoperatively he was able to be up and around with the assist system inactive. Comparison of preoperative and postoperative hemodynamic and roentogenographic studies provided objective evidence of improvement in ventricular function.

The patient was discharged to his home 5.5 wk after operation. His status was relatively stable for 2 wk; readmission for management of empyema then became necessary. Intensive antibiotic therapy led to acute renal failure, and the patient died 96 days after implantation of the partial artificial heart. Autopsy disclosed extensive intrathoracic and abdominal infection, possibly derived from the dehiscence of the thoracotomy wound or from diffusion of oxygen from the prosthesis into the periaortic space. The patch booster itself was intact. Its intravascular surface was covered by a firm fibrin layer and was not involved in the infectious process. There was no evidence anywhere of thromboembolization.

Several conclusions are suggested by this experience:

1. A partial artificial heart located in the descending thoracic aorta and with an intravascular surface of dacron velour backed by a conductive polyurethane may have a very low thromboembolic potential, as compared with prior permanently implanted cardiac assist devices.

2. Intermittent hemodynamic support provided by the dynamic aortic patch enabled a previously bedridden patient with medically intractable left ventricular failure to recover from a major thoracic operation and to achieve substantial improvement in functional capacity.

3. Although improvements in the surgical procedure, the prosthesis, and the driving system are desirable, the present techniques, apparatus, and hardware are practicable for use both in the hospital and at home; a vest-mounted system utilizing rechargeable batteries is probably adequate for short periods outside the home.

4. The dynamic aortic patch provides a promising modality for the rehabilitation of some patients with medically intractable, progressive left ventricular failure. Further investigations of this partial artificial heart are desirable.

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